MEASUREMENT OF STROKE VOLUME BY THE VIBROCARDIOGRAM

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ABSTRACT

An examination has been made of the relationship between stroke volume measured by dye dilution and left ventricular isovolumetric contraction and ejection times as measured by the vibrocardiogram. These studies were performed in 10 normal subjects under postural alterations, and in 11 patients recovering from acute myocardial infarction. Regression analyses of the data showed that ejection time was highly correlated with stroke volume ($r = 0.84$), while use of both ejection time and isovolumetric contraction time improved correlation to $r = 0.90$. Similar correlations were obtained comparing changes in stroke volume with changes in the intervals. It was concluded that the VbCg provides a simple, non-traumatic method for the estimation of stroke volume.

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The need for a simple, non-traumatic technique to measure stroke volume and cardiac output has been greatly intensified by the aerospace program. The physical limitations of devices to be used in the in-flight environment preclude the use of direct cardiac output procedures, such as the indicator dilution and Fick techniques.

Our method for determining stroke volume based on the left ventricular isometric contraction and ejection times in dogs was reported in 1965 [1]. A subsequent comparison with dye dilution studies in human subjects at cardiac catheterization and in dogs using aortic flowmeters confirmed that the ratio of these cardiac phases, as measured from pressure curves, provided a means of quantitating stroke volume [2]. The present study compares stroke volume measured by the dye dilution technique with left ventricular isovolumetric contraction and ejection times determined from the vibrocardiogram [3, 4]. Ten normal subjects were examined under conditions of

postural alteration of stroke volume. Eleven patients recovering from acute myocardial infarction were also studied.

Methods

The normal group consisted of 10 subjects ranging in age from 22 to 28 years. They were classified as normal by negative history, heart auscultation, normal blood pressure and ECG. The 11 patients in the myocardial infarction group ranged from 33 to 83 years and were selected on the basis of sinus rhythm and absence of valvular insufficiency (substantiated by normal dye dilution curves and no murmurs). All had positive evidence of acute infarction by characteristic ECG changes and enzyme elevations.

Cardiac outputs were obtained by the dye dilution technique using cardio-green dye. A Gilford Densitometer and CEC oscillographic recorder were used to transcribe the curves and a modified Stuart-Hamilton formula used for computation.

In the normal group, cardiac outputs were measured during postural change from the supine to the standing position and following return to recumbency. A minimum of three minutes elapsed between the postural change and cardiac output determination. In 5 subjects additional measurements were made in the standing and recumbent positions following the application of leg tourniquets inflated to 80 mm Hg. In the myocardial group all measurements were performed in the recumbent position.

The vibrocardiogram (VbCg) was obtained in the normal group with a crystal microphone* placed at the left parasternal area [5]. This instrument was used in conjunction with a high input impedence coupling amplifier. The VbCg tracing with a Lead I electrocardiogram were simultaneously traced on an Electronics for Medicine recorder using a paper speed of 200 mm/sec. An LTV transducer** and a Honeywell recorder were

* Starling Corporation, Box M, Beverly Hills, California

** Ling, Temco, Vought, Incorporated, Anaheim, California

used in the myocardial infarction group.

Figure 1 shows a typical VbCg curve. The isovolumetric contraction time (ICT) was measured from the peak of the R wave of the ECG to the peak of the J_2 wave of the VbCg. The ejection time (ET) was measured from the J_2 wave to the L wave of the VbCg. Measurements from three cardiac cycles were averaged in each instance.

Results

A total of 76 output determinations were obtained in 21 subjects. Fifty of these were made in the 10 normal subjects and 26 in the 11 subjects with myocardial infarction. These data are given in Tables I and II.

In the normal group, SV's ranged from 29 to 135 ml (mean = 61 ± 20), ICT's from 47 to 100 msec (mean = 72 ± 16), and ET's from 140 to 320 msec (mean = 251 ± 42). In the myocardial infarction subjects, SV's ranged from 19 to 54 ml (mean = 38 ± 8), ICT's from 49 to 91 msec (mean = 71 ± 11) and ET's from 143 to 241 msec (mean = 176 ± 24).

Since the myocardial infarction group data and the normal group data were comparable in their relationship to stroke volume, both groups were treated together in the regression analyses presented. Table III is a summary of these regression data.

Figure 2 illustrates VbCg tracings in two subjects and shows the stroke volume alterations with corresponding changes in ET and ICT.

Figure 3-a compares ICT with SV. An inverse relationship of only minimal significance ($r = 0.43$) was found. ET, however, was highly correlated with SV ($r = 0.83$) (Figure 3-b). Application of regression analysis to the systolic intervals in linear combination improved the correlation coefficient to 0.90 while the ratio, ET/ICT, diminished it to 0.77.

A comparison in the changes (Δ) in the systolic intervals and changes in SV are shown in Figure 4-a and Figure 4-b. There was considerable improvement in the correlation between ICT and SV while the correlation of ET with SV was not improved. The regression of both ΔICT and ΔET on ΔSV gave much the

same result as the absolute duration of these intervals: $r = 0.86$ for ΔET and ΔICT in linear combination and $r = 0.83$ for their ratio.

Figures 5-a and 5-b compare the actual SV's and the predicted SV's using the linear regression formulae. There is uniform scatter about the line of identity with both the absolute data and the deltas, thus justifying the use of linear terms.

Discussion

The relationship between systolic phases and stroke volume has been recognized for some time. The early investigations of Frank [6], Wiggers [11], and Remington, et al [9], and the more recent studies of Jones and Foster [7], and Weissler, et al [10], have shown that the ejection interval is directly influenced by stroke volume, if myocardial contractility remains unchanged. These observations are confirmed by the high correlation between stroke volume and the ejection period found in this study. ICT, because of its inverse relationship to the rate of ventricular pressure rise,

was used in conjunction with ET to compensate for any contractility alterations. Such a compensation was found in previous work to improve markedly the correlation between stroke volume and ejection time [2]. That only minimal improvements were obtained by inclusion of the isometric interval in this study indicates that cardiac contractility was not greatly altered by postural changes.

Two potential sources of error may be present in the SV-interval correlation in addition to measurement error. First, the dye-output technique has at best a 90% compatibility with the direct Fick [8], and may itself account for some of the scatter in the data. Second, a non-uniform relationship of the intervals to SV may exist between individuals. Comparisons of actual and predicted SV data from 4 subjects in whom sufficient alterations occurred to permit individual analysis are shown in Figure 7. These indicate that the correlation in the individual is much higher than in the group. It is thus likely that the interval-SV relationship is modified by a factor not measured in this study.

It should be stressed that these results were obtained from normal subjects undergoing postural alterations and from supine patients during recovery from myocardial infarction. It is not known whether the same regression formulae can be applied to other conditions. It should also be noted that the relationship of the systolic intervals to stroke volume applies only to conditions of true isovolumetric contraction in the absence of significant pressure gradient, and to those instances where the forward stroke volume represents the total ventricular output. Nevertheless, within the limitations stated, the vibrocardiographic technique provides a method for quantitatively estimating stroke volume and for determining its directional changes.

Summary

An examination has been made of the relationship between stroke volume measured by dye dilution and left ventricular isovolumetric contraction and ejection times as measured by the vibrocardiogram. These studies were performed in 10 normal subjects undergoing postural alterations, and in 11 patients recovering from acute myocardial infarction. Regression

analyses of the data showed that ejection time was highly correlated with stroke volume ($r = 0.84$), while use of both ejection time and isovolumetric contraction time improved correlation to $r = 0.90$. Similar correlations were obtained comparing changes in stroke volume with changes in these intervals. It was concluded that within the limitations described the VbCg provides a simple, non-traumatic method for the estimation of stroke volume.

*Footnote

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Subject	Age	Position	SV	ICT	ET	Ratio	HR
1	22	S	49	100	225	2.2	86
		RT	52	85	262	3.1	62
		R	86	59	296	5.0	60
2	22	R	84	58	270	4.7	79
		S	44	57	202	3.0	113
		ST	47	75	217	2.9	105
		RT	78	62	265	4.3	80
		R	88	60	273	4.5	74
3	21	R	84	50	300	6.0	72
		R	77	52	307	5.9	72
		S	56	74	232	3.1	84
		ST	50	70	222	3.2	84
		ST	51	74	233	3.1	84
		RT	82	59	277	4.7	72
		R	101	55	287	5.2	72
4	22	R	90	55	300	5.5	75
		S	59	65	250	3.8	96
		ST	46	58	252	4.3	96
		RT	68	60	302	5.0	72
		R	79	58	300	5.2	74
5	28	R	74	55	308	5.6	61
		S	34	86	215	2.5	95
		ST	29	86	205	2.4	102
		RT	69	79	290	3.7	57
		R	68	80	297	3.7	57
6	19	R	47	71	283	4.0	72
		S	44	71	256	4.0	84
		ST	50	71	250	3.5	78
		RT	51	82	282	3.4	60
		R	59	74	287	3.9	66
7	24	R	70	80	250	3.1	86
		R	68	77	250	3.2	88
		S	38	95	175	1.8	120
		S	38	95	175	1.8	110
		ST	30	100	140	1.4	127
		ST	33	95	150	1.6	127
		RT	50	100	200	2.0	97
		RT	54	105	210	2.0	91
		R	57	95	250	2.6	88
		R	65	95	250	2.6	82
8	33	R	71	50	285	5.7	84
		S	54	80	225	2.8	96
		ST	47	83	215	2.6	96
		R	73	62	272	4.4	84
9	23	R	62	52	282	5.4	80
		S	58	47	254	5.4	84
		ST	55	47	260	5.5	80
		RT	43	56	231	4.1	91
		R	44	55	214	3.8	97
17	21	R	135	58	320	5.5	60

KEY

S = Standing
R = Recumbent
ST = Standing with Tourniquet
RT = Recumbent with Tourniquet
SV = Stroke Volume determined by dye dilution
ICT = Isovolumetric Contraction Time measured from R-J_2
ET = Ejection Time measured from J_2-L

Table I

Subject	Age	SV	ICT	ET	Ratio	HR
10	73	48	82	185	2.3	86
		51	80	200	2.5	88
11	33	54	60	210	3.5	113
		52	60	180	3.0	113
		51	50	170	3.4	120
		39	80	170	2.4	82
12	83	47	49	196	3.9	94
		36	82	192	2.4	83
		37	63	220	3.5	88
		36	82	192	2.3	83
13	77	32	68	165	2.4	97
		28	69	150	2.2	141
14	54	31	72	178	2.5	86
		30	75	147	2.0	80
15	68	30	91	165	1.8	79
		41	63	170	2.7	90
		36	75	179	2.4	85
		28	53	143	2.7	107
16	60	30	75	150	2.0	110
		40	72	178	2.5	105
		39	72	169	2.3	116
		45	71	169	2.4	115
18	73	19	85	144	1.7	128
19	50	36	82	165	2.0	89
20	66	41	78	241	3.1	85
21	69	37	63	145	2.3	89

Table II

SV (± 18) = -0.66 (ICT) + 100.5	r = 0.43
SV (± 11) = 0.32 (ET) - 19.9	r = 0.83
SV (± 9) = -0.48 (ICT) + 0.31 (ET) + 16.7	r = 0.90
ΔSV (± 13) = 0.75 (ΔICT) + 0.75	r = 0.61
ΔSV (± 9) = 0.30 (ΔET) + 0.63	r = 0.84
ΔSV (± 8) = -0.28 (ΔICT) + 0.33 (ΔET) + 0.60	r = 0.86

SV = ml

ICT and ET = msec

ΔSV = Change in SV

Table III

LEGEND

Figure 1 Methods of measuring cardiac phases from the VbCg (top trace) and ECG. ICT = Isovolumetric Contraction Time ET = Ejection Time

Figure 2 Representative tracings from two subjects undergoing postural changes. Note the diminished ET and prolonged ICT occurring with the reduced stroke volume in the standing position.

Figure 3 a) ICT was inversely related to SV, but with minimal significance.

b) A highly significant relationship was found with the ejection interval and stroke volume.

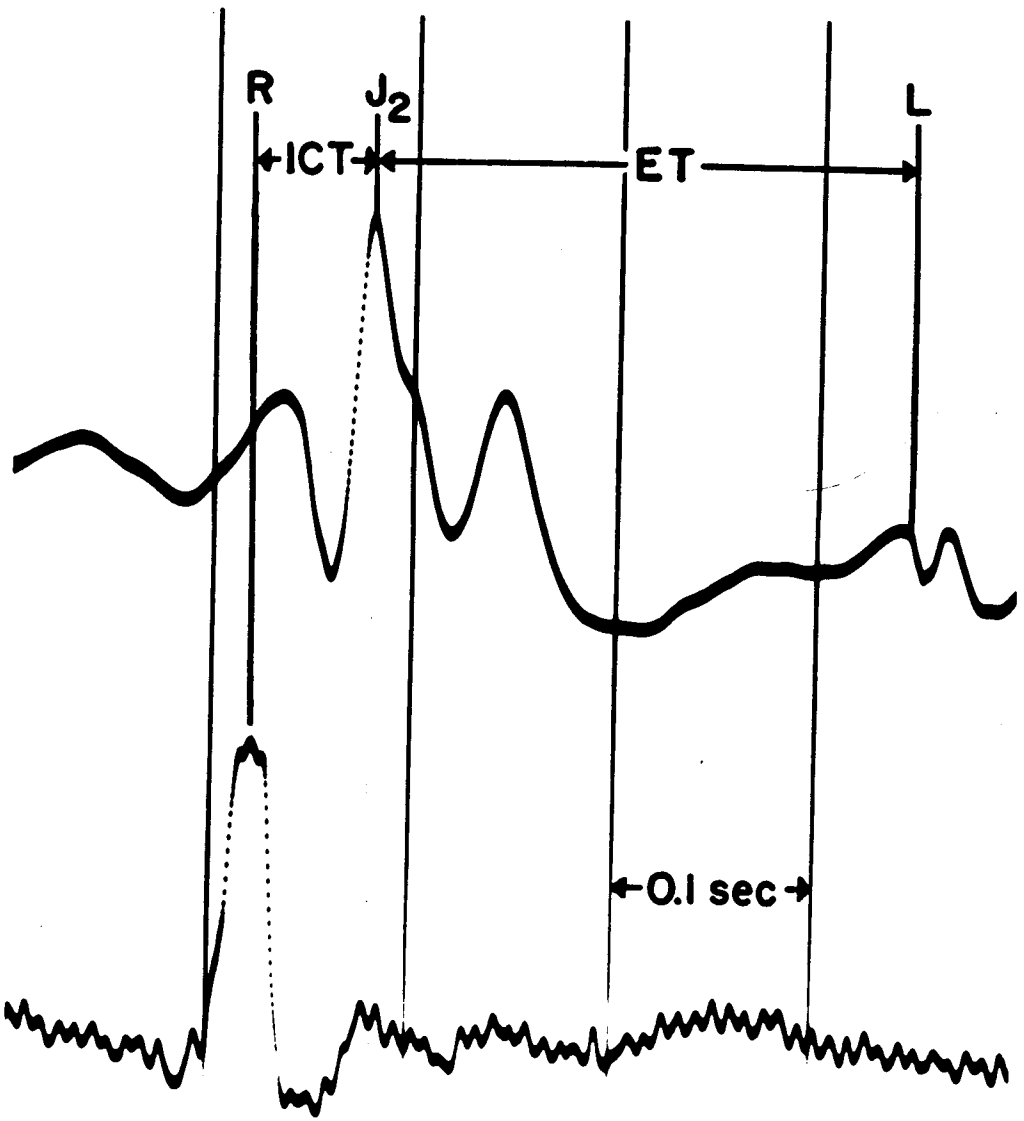
Figure 4 a) The change in ICT showed a higher inverse correlation with the change in SV than did the duration of ICT with SV.

b) The change in ET showed equal correlation with the change in SV as did the duration of ET with SV.

Figure 5 a) Predicted stroke volumes obtained from the regression formula compared with actual stroke volumes. Note that most values fall within ± 20%.

b) The predicted change in stroke volume compared with the actual change in stroke volume. There is less scatter in the data than with absolute value of SV.

Figure 6 Comparison of predicted SV obtained from the regression formula and actual SV in four subjects. Note the high correlation but with a slightly different slope in each individual.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L
ICT
ET
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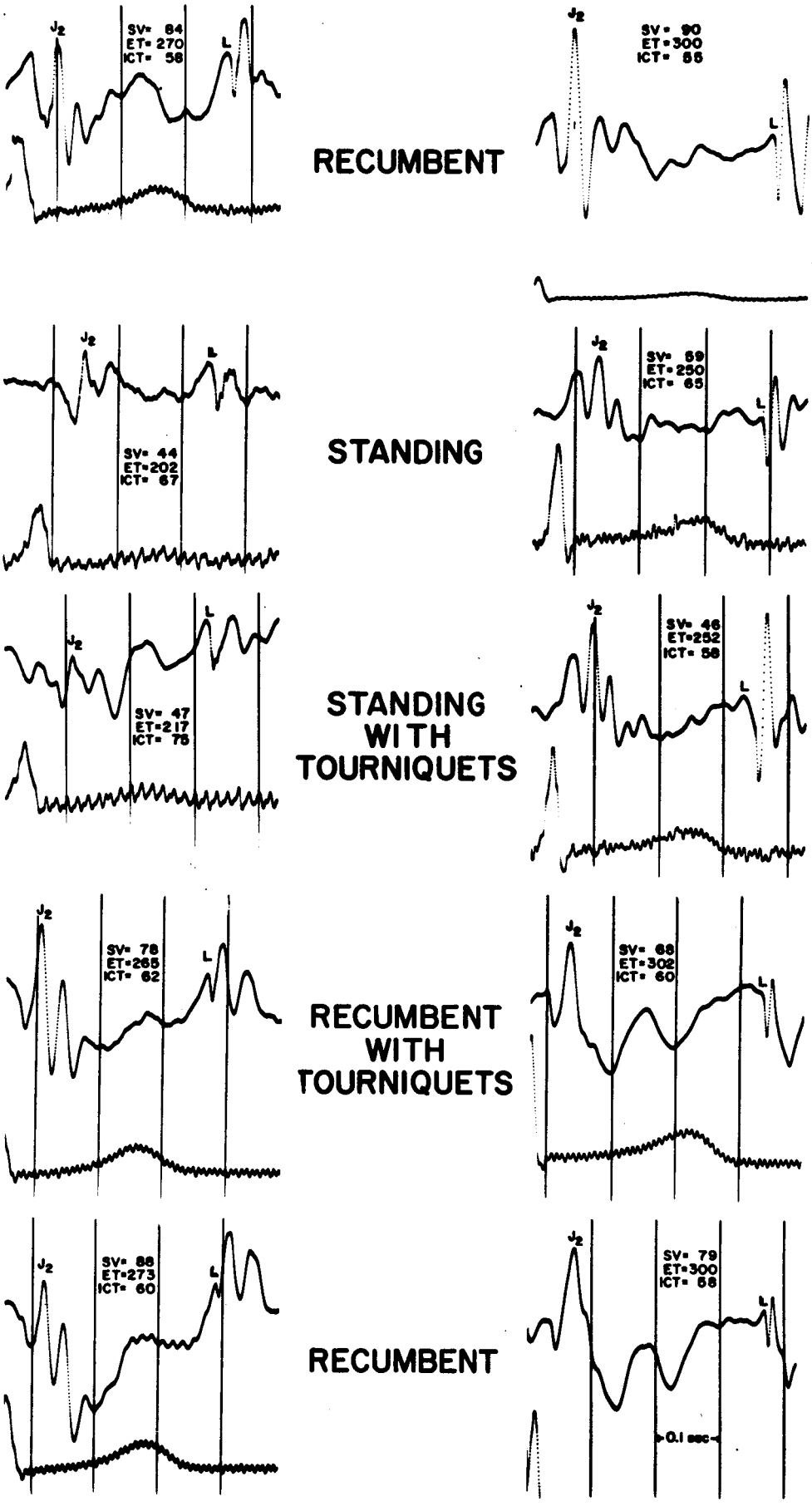
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ICT= 58
L
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J2
SV= 90
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J2
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ET=202
ICT= 67
STANDING
J2
SV= 59
ET=250
ICT= 65
L
L
J2
SV= 47
ET=217
ICT= 75
STANDING
WITH
TOURNIQUETS
J2
SV= 46
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J2
SV= 78
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ICT= 62
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WITH
TOURNIQUETS
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ET=302
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L
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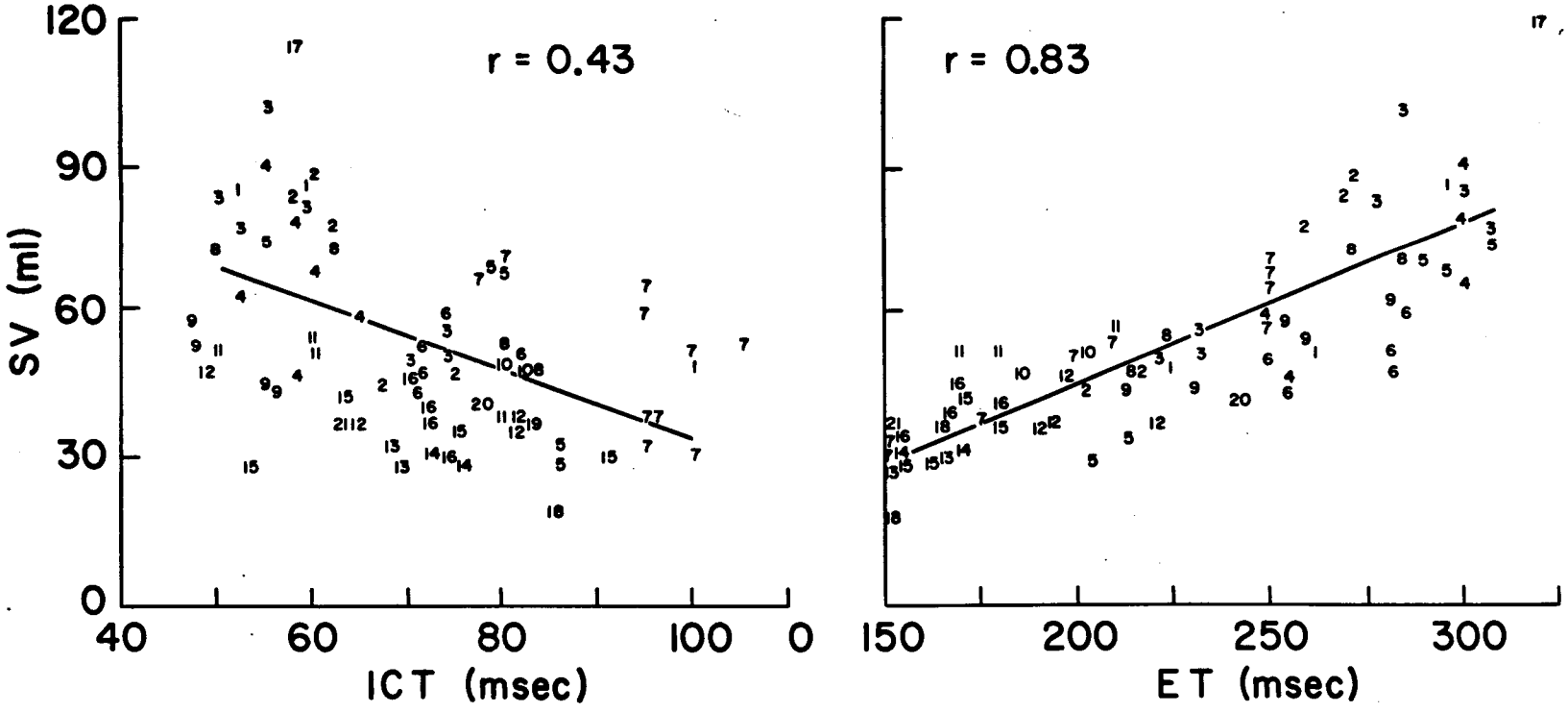
L
0.1 sec

Figure 2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SV (ml)
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90
60
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40
60
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100
0
ICT (msec)
r = 0.83
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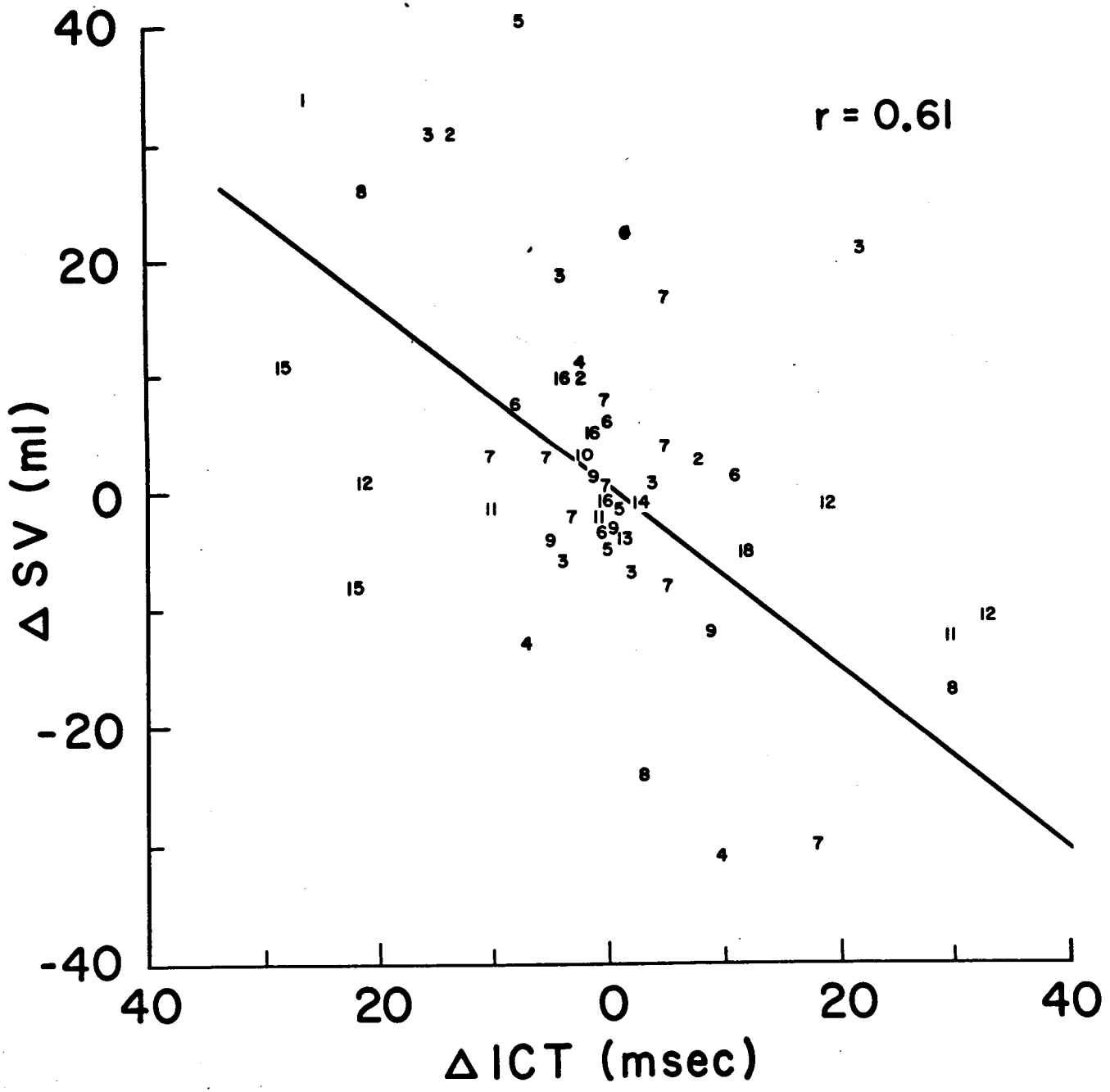
200
250
300
ET (msec)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Δ ICT (msec)
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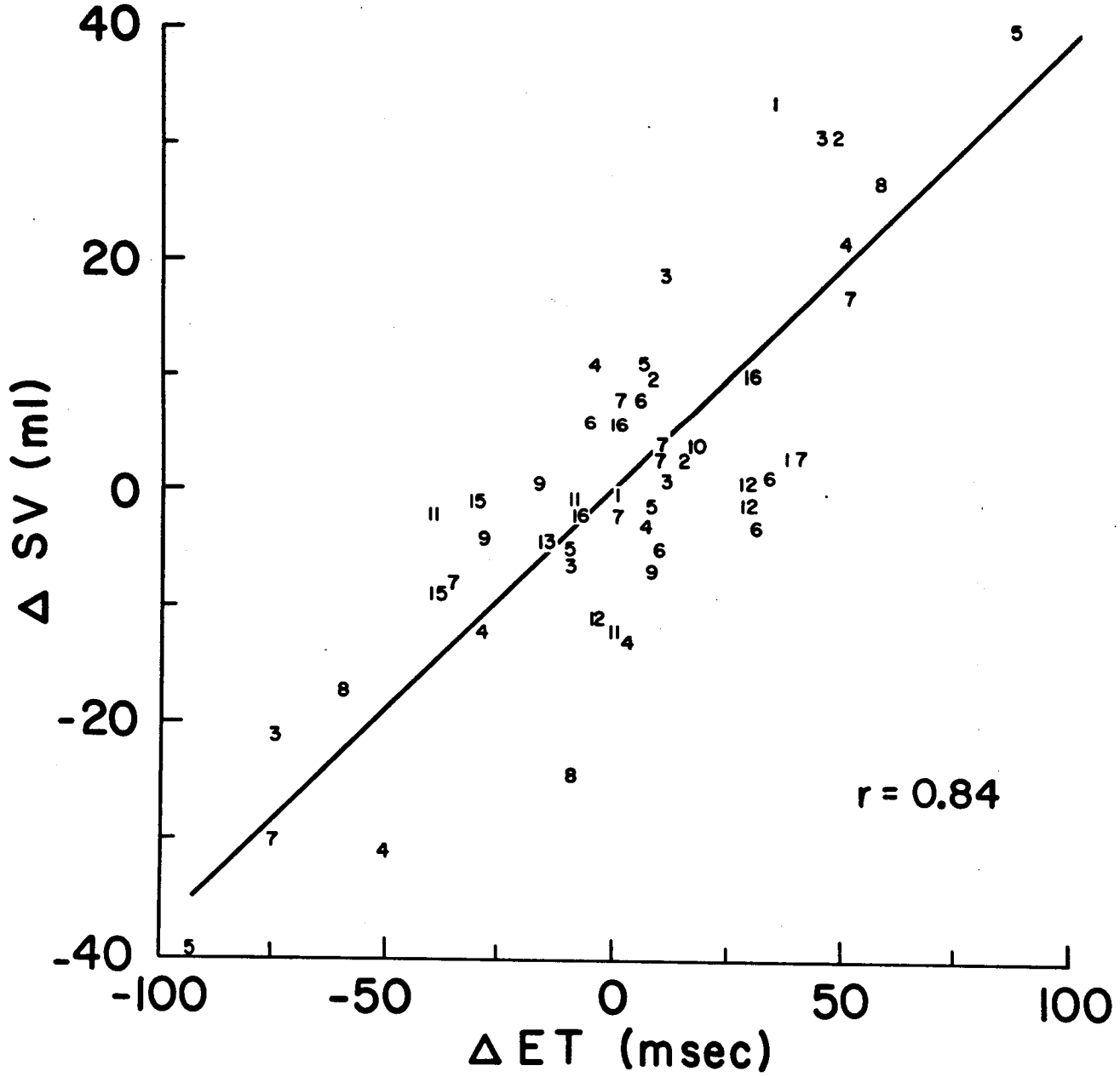
20
0
20
40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50
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Δ ET (msec)
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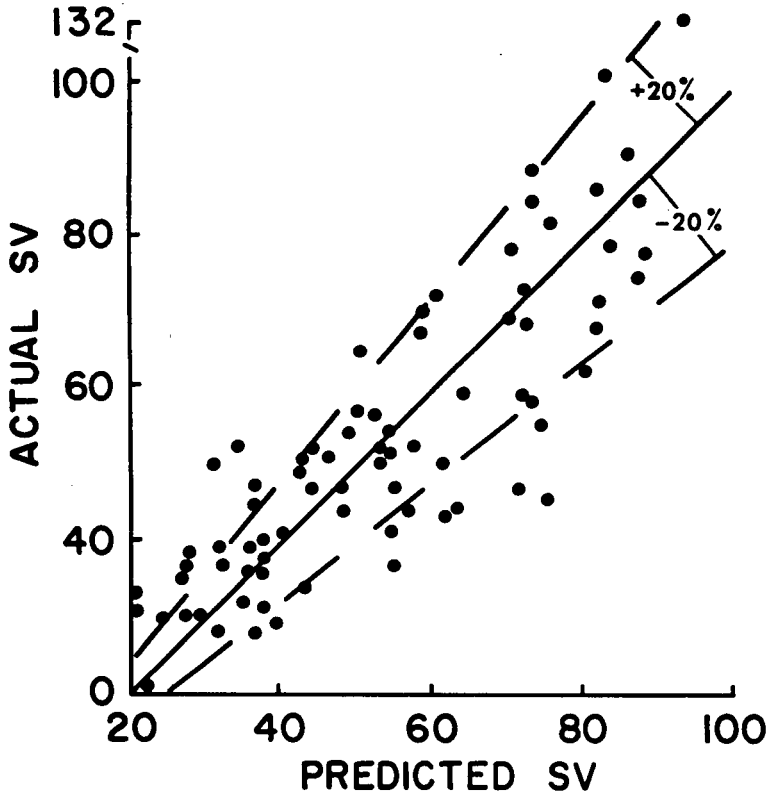
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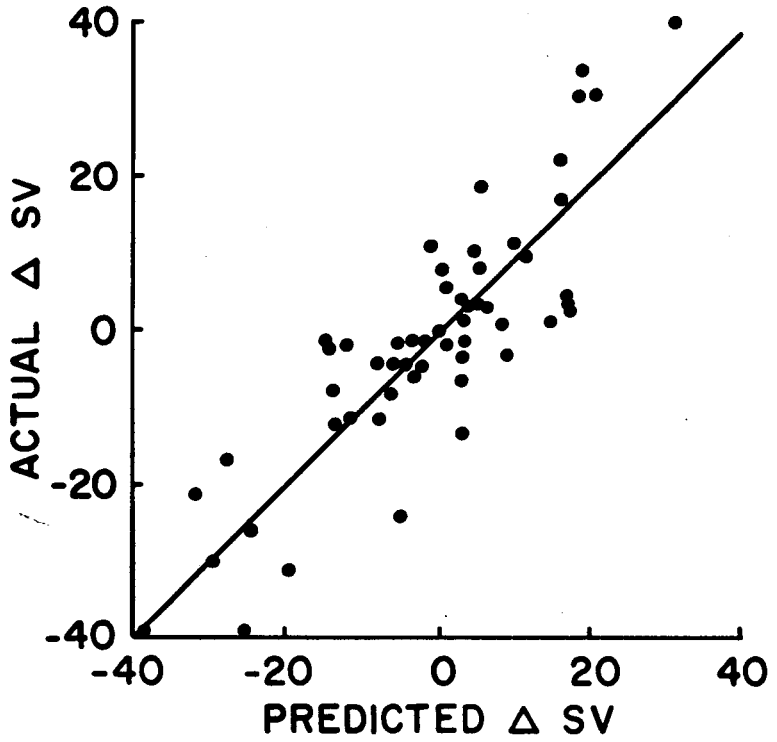
PREDICTED SV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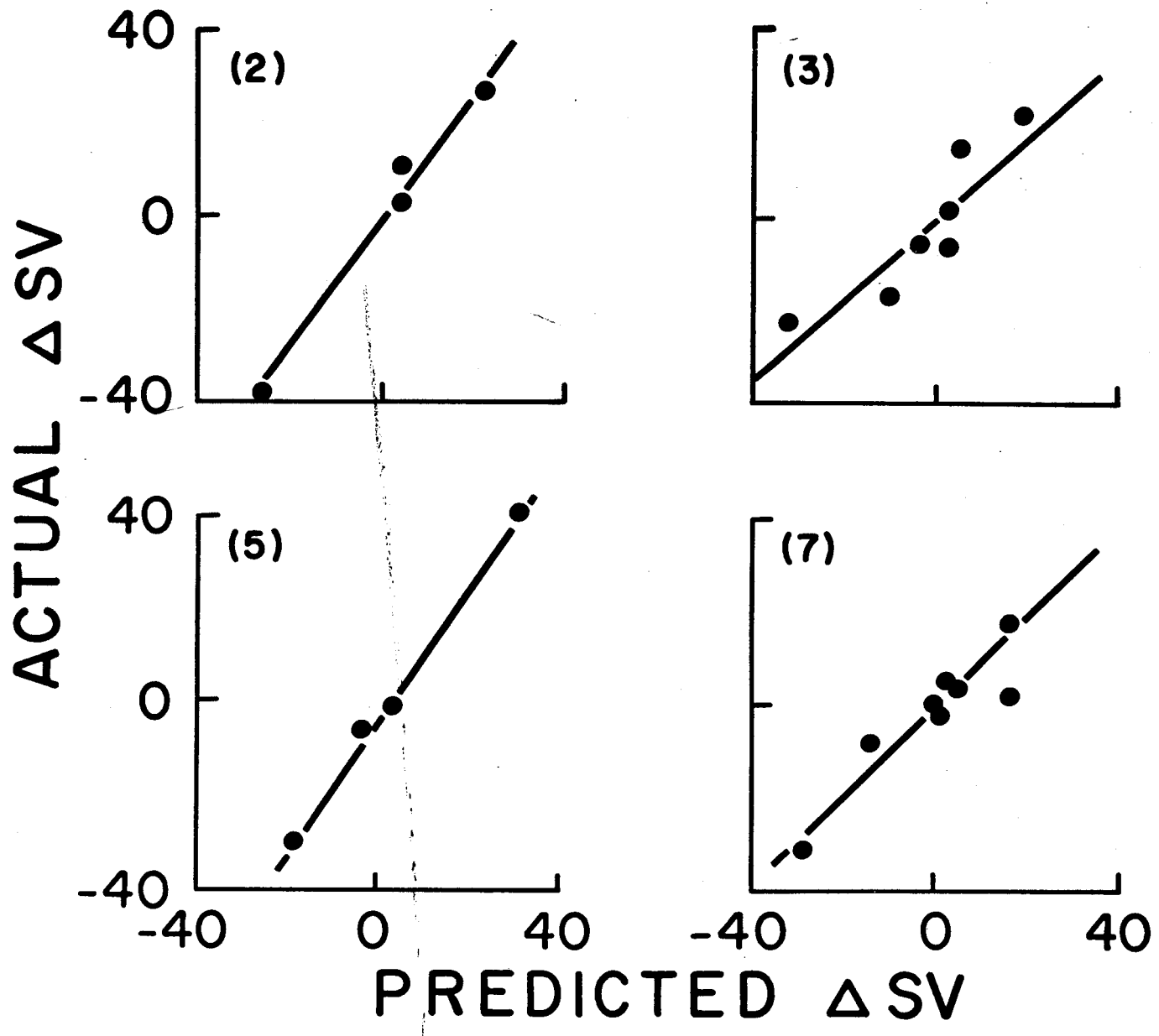
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(3)
(5)
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ACTUAL Δ SV
PREDICTED Δ SV